THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

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RELATIVE INCIDENCE OF INTERNAL CORK OF SWEETPOTATOES IN OKLAHOMA IN 1950 AND 1951

F. Ben Struble, H. B. Cordner, and Lou S. Morrison

Internal cork (virus) of sweetpotatoes was first reported by Nusbaum (1) in South Carolina. Since then this disease has been found in Georgia, North Carolina, Virginia, Tennessee, Mississippi, Maryland, Louisiana, Alabama, and Texas (Nusbaum, 2).

Cork was found for the first time in Oklahoma in the fall of 1949. A survey made in 1946 by Dr. D. A. Preston, formerly of the Department of Botany and Plant Pathology, failed to reveal the presence of internal cork in the sweetpotato stocks of certified plant growers and several commercial growers in Oklahoma.

Following the discovery of cork in 1949, a survey was made to determine the extent and relative severity of the disease in the State. This survey was made January 12-15, 1950. Storage houses of 12 different growers at widely scattered locations over the State were visited and samples of sweetpotato seed stocks examined. Each root of each sample was sliced with a power-driven slicing machine; and the individual slices, each approximately 1/10 inch thick, were observed for cork. Although the number of roots varied from sample to sample, an effort was made to have about 30 roots in each sample. Twenty-two different sweetpotato varieties and seedlings were examined in the 1950 survey.

Internal cork was found in all except one of the areas surveyed in 1950. At five locations the disease was not found in all the sweetpotato varieties sampled. No one variety, however, was found consistently free of cork in all locations. In general, when cork was found it was present in only trace quantities or less as determined by the scale used by Nusbaum (2). The range on this scale is from 0 (no cork) to 4 (severe) and the various ratings are determined by the relative size of the cork spots or aggregates of spots. Cork was found most consistently and to a greater extent in the variety Porto Rico and derivatives from it, especially bunch types.

In 1951, during the period of February 19-22, a follow-up survey was made to determine if internal cork was increasing in severity. In this survey sweetpotatoes were examined from the storage houses of 14 different growers, in most cases the same growers whose stocks were sampled in 1950. Twenty-four different sweetpotato varieties and seedlings were sampled. Methods of sampling and slicing the roots were the same as those used in 1950.

During the 1951 survey, cork was found in all areas surveyed, but again was not found in all varieties at all locations. In only one instance, a sample of Bunch Porto Rico, was cork found in quantities greater than a trace. Comparative data for incidence of cork in 1950 and 1951 are shown in Table 1. The data presented here for the two years are comparable in that the roots sliced in 1951 were grown from seed stock which had been sampled in 1950. Any data from

Table 1. Comparative incidence of internal cork in sweetpotatoes in 1950 and 1951.

Location and sweetpotato variety or seedling	1950			1951		
	Roots sliced	Roots with cork	Cork spots per infected root	Roots sliced	Roots with cork	Cork spots per infected root
	No.	percent	Av. No.	No.	percent	Av. No.
Hennessey						
Allgold	12	8.3	1.0	30	20.0	1.1
Isabella						
Allgold	30	0.0	0.0	20	5.0	1.0
Thomas						
Porto Rico	26	3.8	1.0	30	16.5	1.8
Allgold	20	20.0	1.0	30	20.0	1.1
Okla. 17	20	5.0	1.0	30	16.5	1.0
Okla. Golden	29	17.2	1.4	20	5.0	1.0
Rush Springs						
Location A.						
Porto Rico	4	25.0	1.0	15	26.4	1.0
Yellow Jersey	31	19.3	1.1	28	10.5	1.0

Table 1. Continued

Location and sweetpotato variety or seedling	1950			1951		
	Roots sliced	Roots with cork	Cork spots per infected root	Roots sliced	Roots with cork	Cork spots per infected root
	No.	percent	Av. No.	No.	percent	Av. No.
Rush Springs						
Location B						
Porto Rico	24	12.5	1.0	30	6.6	1.5
Improved Porto Rico	22	9.0	1.0	30	3.3	1.0
Golden (Porto Rico)	29	0.0	0.0	30	9.9	1.0
Ringling						
Porto Rico	27	7.4	1.0	25	8.0	2.5
Ardmore						
Allgold	9	0.0	0.0	30	0.0	0.0
Improved Porto Rico	30	10.0	1.0	23	8.6	1.0
Idabel						
Location A						
31x36-3	13	0.0	0.0	22	9.0	1.0
Okla. 7	13	15.3	1.0	30	20.0	1.0
Okla. 13	15	0.0	0.0	20	20.0	1.0
Okla. 17	15	6.6	1.0	30	3.3	1.0
Okla. 26	17	23.5	1.0	30	13.3	1.0
Okla. 38	20	10.0	1.0	30	0.0	0.0
Improved Porto Rico	15	6.6	1.0	30	16.5	1.2
Rols	15	0.0	0.0	29	3.4	1.0
Allgold	29	20.6	1.0	15	6.6	2.0
Idabel						
Location B						
Porto Rico	22	9.0	1.0	19	20.8	1.0
Improved Porto Rico	28	10.7	1.0	22	18.0	1.0
Okla. 17	30	10.0	1.0	15	13.2	1.0
Okemah						
Improved Porto Rico (draws)	30	16.6	1.4	27	3.7	1.0
Improved Porto Rico (vine cuttings)	28	10.7	1.0	30	3.3	1.0
Okla. 17	25	4.0	1.0	30	6.6	1.0
Allgold (vinecuttings)	30	0.0	0.0	30	0.0	0.0

samples not having a continuous seed stock history have been omitted from this Table. Unless otherwise indicated each variety was propagated from draws. Some growers propagate from vine cuttings to secure nematode-free seed stock.

Figure 1 shows the areas surveyed. Since internal cork has been found in each of these areas, the disease apparently is widespread in the State.

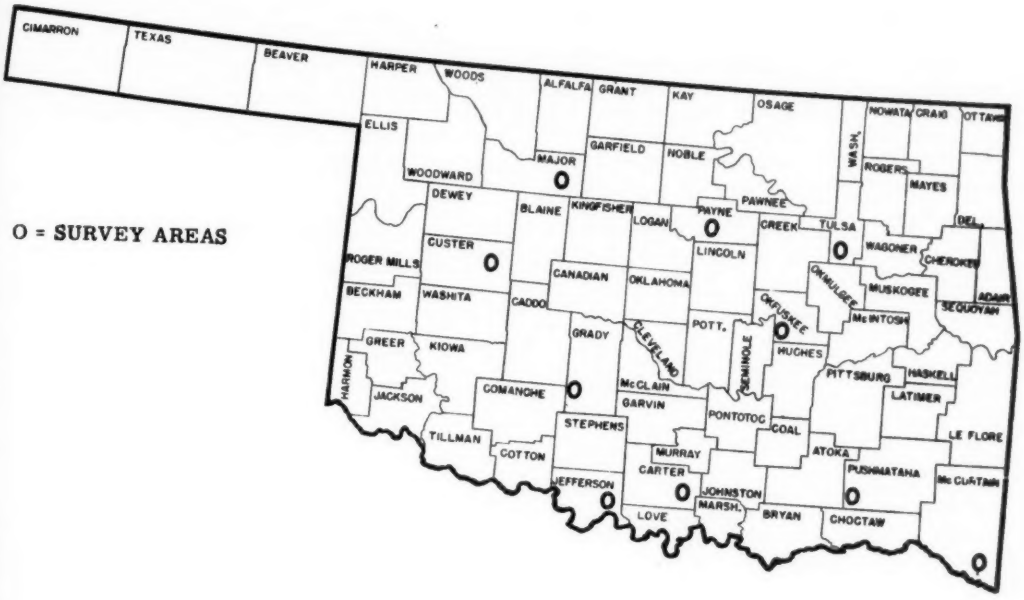

FIGURE 1. Areas in Oklahoma surveyed for internal cork of sweetpotatoes. Cork was found in every area surveyed.

The data in Table 1 indicate no consistent trend so far as increase or decrease of internal cork is concerned. In 14 of the samples there was a definite increase for 1951 in the percentage of sliced roots showing cork. In 10 of the samples there was a definite decrease, and in 6 samples there was no significant change. The increase or decrease of cork was not consistently associated with any one variety. The over-all impression obtained from the 1951 survey was that the cork situation was not materially worse in 1951 than it had been in 1950. On the basis of two years' observations it is our opinion that internal cork is at present of relatively minor importance in sweetpotatoes in Oklahoma.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY AND DEPARTMENT OF HORTICULTURE
OKLAHOMA AGRICULTURAL EXPERIMENT STATION, STILLWATER, OKLAHOMA

SWEETPOTATO INTERNAL CORK IN CALIFORNIA

R. G. Grogan and G. C. Hanna

Internal cork of sweetpotato, which has not been reported previously from California, was found during the summer of 1950 in varietal trial plots located at Davis and Shafter and in an experimental planting of Bunch Porto Rico at Manteca. Several root samples suspected of having the disease were sent to Dr. C. J. Nusbaum who confirmed the diagnosis. [1]

A list of the varieties which were planted in the plot at Davis, with notations as to the presence or absence of internal cork is presented in Table 1.

Table 1. Sweetpotato varieties grown at Davis, California in 1950 with notes on source of seed and occurrence of internal cork.

Variety	Source of seed	Date seed obtained	Internal cork %
Ranger	U. S. D. A.	1946	0
LI38 (Heartogold)	"	1948	10
B5941	"	"	0
B5944	"	"	0
B5999	"	"	0
Bunch Porto Rico (Georgia bunch)	"	"	50
Oklahoma 24 (Allgold)	"	1949	3
Improved Red Porto Rico	Manteca, California	1948	50
Velvet Porto Rico[a]	"	"	4 ?
Murft Bunch Porto Rico (Texas 51)	Gilmer Station, Texas	1950	1
Texas Improved Porto Rico	"	"	0
Golden Skin	"	"	0
Red Jersey[b]	Stockton, California	"	0
Red Nancy Hall[b]	Kansas	"	0
Rols[b]	"	"	0
Orlis[b]	"	"	0

[a]Brown sunken spots which were not delimited by a phellum layer were found near the edge of a few roots of this variety. No conclusions concerning the cause of this symptom have been reached.
[b]Stocks of these varieties were limited and only a few roots of each have been examined.

All determinations were made by cutting thin slices of approximately 500 roots which had been cured and stored for two or more months. The leaf symptoms which have been reported[2] as characteristic for this disease were not found in any of the field plantings. However, Dr. W. C. Snyder observed ringspot and mottle symptoms on the leaves of Bunch Porto Rico slips grown from diseased roots in a greenhouse at Berkeley.

All of the varieties listed in Table 1 have been grown in close proximity to the two diseased varieties, Bunch Porto Rico and Improved Red Porto Rico, during the 1950 season and all that were received in 1946 or 1948 have been grown in a plot with the diseased varieties each season since 1948. Bunch Porto Rico has been used in guard rows between replications and therefore occurred in every fourth row throughout the plots.

Since the planting stocks were not examined for internal cork symptoms when recieved, it is not known whether the disease has spread from some varieties to others or whether all varieties which now show symptoms were diseased when received. However, if spread has occurred, it has been extremely erratic, since several of the varieties which have had ample opportunity to

[1]The cooperation of C. J. Nusbaum of the University of North Carolina is gratefully acknowledged.
[2]Nusbaum, C. J. Internal cork of sweetpotatoes. South Carolina Agr. Expt. Sta. Bull. 381: 1-23, 1950.

contract the disease were apparently unaffected at the end of the 1950 season.

A survey is being conducted by the Division of Plant Pathology of the State Department of Agriculture to determine if commercial seed stocks of Improved Red Porto Rico and other varieties that are widely grown in the State are infected with internal cork.

DIVISIONS OF PLANT PATHOLOGY AND TRUCK CROPS, UNIVERSITY OF CALIFORNIA AGRICULTURAL EXPERIMENT STATION, DAVIS, CALIFORNIA

CURLY-TOP ON WILD SEEDLING POTATOES

R. D. Watson and T. E. Randall

In June 1950 a collection of 11 types of wild potato seedlings was exposed in the curly-top field plots to determine their reaction to the curly-top virus. [1] The seed came from collections made in Mexico and the southwestern United States. Their sources and some information concerning the potatoes used in these tests are given in Table 1. P-1 to P-7 were received from Dr. Donald Reddick of Cornell University, Ithaca, New York. P-8 to P-14 were received from R. W. Hougas, Potato Introduction Station, Department of Genetics, University of Wisconsin, Madison, Wisconsin. A brief statement of resistances to other diseases and insects is included. It was hoped that these collections were from an area in which curly-top had been a prevalent disease, and that the species through natural selection and survival would be resistant to it.

Table 1. Potato seedlings used in curly-top eliminations.

P1 -- Solanum antipoviczii, collected by D. S. Correll, October 23, 1947 Molina de Fores Est. Mexico, Mexico. Heterozygous for late blight immunity.
P3 -- S. antipoviczii or S. neo-antipoviczii, collected by D. S. Correll, Nov. 8, 1947 in Mexico. Myzus persicae die on this host; it is late-blight susceptible.
P5 -- S. species, mixed S. simplicifolium and S. antipoviczii, collected by D. S. Correll, Oct. 31, 1947 in Mexico.
P6 -- S. demissum, late-blight immune. The typical green stem demissum. Collected by D. S. Correll Nov. 15, 1947 at Villa Hermosa Nevada de Toluca, Est. Mexico.
P7 -- S. polyadenium, late-blight immune, resistant to most potato insects. Collected by D. S. Correll, Dec. 23, 1947. Matujeo Michoacan, Mexico.
P8 -- S. andigenum or tuberosum, Mexico 161774.
P9 -- Solanum species resistant to late blight. Mexican Potato 161770.
P10 -- S. polyadenium resistant to insects and late blight Mexican 161728.
P11 -- S. andigenum or tuberosum. Mexican potato 161350.
P12 -- S. andigenum or tuberosum. Mexcian potato 161285.
P14 -- S. species, Mexican potato. 160226.

Commercial potatoes are sometimes infected with curly-top virus, but usually show no evident field symptoms. This failure to show symptoms may be due to resistance or to the rapid early growth made by these tuber-planted potatoes. For this reason seedling transplants were placed in the field and were treated in such a way as to bring out the maximum symptom expression.

The plants were pruned by removing most of the top growth on August 11 to force new growth and to give the leafhoppers a better chance to inoculate them. The effectiveness of the treatment is indicated by the large number of plants showing clear-cut symptoms (Table 2) following this type of treatment. In Table 2 the relative severity of curly-top in the field is

[1] The plots, located near Moxee City, Washington, are made available through the Washington State Department of Agriculture.

Table 2. Wild potato seedlings showing curly-top under field conditions.

Field number		Number plants	Number plants with curly-top July 20	August 10*	Sept. 14*
P1	Solanum antipoviczii	2			1
P3	S. antipoviczii or neo-antipoviczii	2			2
P5	S. species mixed	2		2	2
P6	S. demissum	2		1	2
P7	S. polyadenium	2			2
P8	S. species 161774	1		1	1
P9	S. species 161770	3			2
P10	S. polyadenium 161728	2		1	2
P11	S. andigenum or S. tuberosum 161350	4			2
P12	S. andigenum or S. tuberosum 161285	1			1
P14	S. species 160226	1			1
Total		22	0	5	18
Percentage of curly-top on commercial tomatoes under similar exposure			82	97	100

* Plants cut back August 11 to force new growth. This made symptoms much more pronounced by September 14.

shown by the amount of curly-top in tomatoes and the effect of topping on bringing out the symptoms on potatoes. Eighteen of the 22 plants showed clear symptoms of curly-top on the new growth by September 14. Those plants not showing evident symptoms were more like S. tuberosum. The new growth on the pruned plants showed symptoms much more clearly than the old growth. At least one or more plants of all the 11 types (species) developed curly-top in the field. This is an example of the value of cutting back the tops to improve the evaluation of curly-top reaction.

COOPERATIVE INVESTIGATION OF THE IDAHO AGRICULTURAL EXPERIMENT STATION, MOSCOW, AND THE WASHINGTON AGRICULTURAL EXPERIMENT STATION, PULLMAN

SOME PRELIMINARY OBSERVATIONS ON CUCURBIT VIRUSES IN FLORIDA

C. W. Anderson

Three viruses or virus strains infectious to cucurbits were collected in central Florida in 1950, as follows: virus 1, collected from cantaloupe, virus 2, from Commelina gigas Small; virus 3, from Crookneck squash. This paper presents preliminary data toward the identification of these viruses.

HOST RANGE: Mechanical transmission of viruses 1 and 3 was attempted to cucumber, Early Prolific Straightneck squash, watermelon, Nicotiana tabacum L., N. glutinosa L., tomato, pepper, beet, celery, Zinnia elegans Jacq., cowpea, Bountiful bean, and broad bean. Virus 1 produced symptoms resembling those of cucumber mosaic strains in all the above species except cowpea and bean, and was recovered from all except cowpea (no recovery was attempted from bean or broad bean). Virus 3 produced symptoms only in squash, cucumber, and watermelon. At least 20 plants of all species not showing symptoms of virus 3 were inoculated, and recovery was attempted from each species except bean and broad bean. The virus was recovered only from the cucurbits. Virus 2 inoculum was introduced into most of the above species, including watermelon, and produced symptoms usually resembling those of virus 1. The two viruses were not identical, as shown by the consistently different symptom reactions on several hosts, but particularly on pepper and tomato.

LONGEVITY IN VITRO: The resistance to aging of viruses 1 and 3 was tested, using squash as both inoculum and test plants. Virus 1 resisted 9 days' aging; virus 3 withstood 7 but not 14 days. Virus 3 was consistently less infectious after 3 or 4 days' aging than virus 1.

ACQUIRED IMMUNITY REACTIONS: Virus 1 produced necrotic primary lesions in watermelon and appeared very slow to produce systemic infection. Virus 3 caused no primary lesions on watermelon. Its incubation period was usually 6 to 14 days. Early symptoms were mild vein clearing, and downward curling of the leaf margins. Later symptoms included reduction of leaf size, some stunting, and mosaic pattern in which dark green blisters or bands often occurred near the veins. When watermelons in the cotyledon stage were inoculated with virus 3 and the first two leaves were later inoculated with virus 1, the number of primary lesions produced by virus 1 was reduced about 88 percent by the presence of virus 3. Experiments to test for possible protection afforded by virus 1 against virus 3 in the cucumber were complicated by three factors, namely: (a) watermelon, the most suitable plant to use in recovery attempts for virus 3, often showed severe cotyledon necrosis produced by virus 1; (b) watermelon also displayed severe stunting and hypocotyl necrosis caused by virus 1; and (c) occasionally virus 1 produced severe systemic symptoms in watermelon as a result of early systemic invasion. However, the distinctive symptoms of virus 3 did appear in a large percentage of the watermelon plants that did not show severe systemic symptoms of virus 1 infection at an early date. In these cases, the incubation period of virus 3 was slightly longer than it was in plants inoculated with virus 3 only, and the third true leaf was usually the first to show symptoms of virus 3 infection rather than the second. The writer hopes to publish a more detailed account of this work at a later date; however, the results show that the presence of virus 1 does not prevent virus 3 from invading cucumber systemically.

CONCLUSIONS: Viruses 1 and 2 are strains of cucumber mosaic. Both are infectious to watermelon and in this resemble the southern celery mosaic strain previously described (Wellman, 6) from Florida. These two strains are not identical, and their occurrence indicates that possibly other strains of cucumber mosaic infectious to watermelon may be present in Florida.

Virus 3 is easily differentiated from the well-known mechanically transmissible viruses infectious to cucurbits by its restricted host range. It differs from cucurbit mosaic (cucumber viruses 3 and 4) described by Ainsworth (2) and from squash mosaic (Freitag, 3) in the short time it remains active in vitro, while it differs from Aycock's (1) cantaloupe mosaic in its symptoms on watermelon. Its symptoms somewhat resemble those of the mosaic of watermelon previously reported from Florida by Walker (5). There is some evidence that virus 3 tends to protect watermelon against the cucumber mosaic strain herein referred to as virus 1. However, failure of the virus 1 strain to protect cucumber from virus 3 indicates that the two viruses are not very close in their relationship, a fact borne out by the wide differences in host range

between the two. Price (4) has shown that reductions of up to 75 percent in the number of lesions are sometimes produced by previous inoculation of a plant with an unrelated virus, but such reductions are rare. The 88 percent reduction in the number of lesions produced by cucumber mosaic in leaves that show no symptoms of virus 3, or vein clearing only, may mean that virus 3 is distantly related to cucumber mosaic. Leaves produced later were not used because they are commonly so stunted and rolled by virus 3 that they may not give results comparable to the controls.

The common name "southern squash mosaic" is suggested for virus 3 until its relationships with other viruses can be more thoroughly worked out. Information concerning the insect transmission and natural sources of southern squash mosaic in the cucurbit fields is needed.

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DEPARTMENT OF PLANT PATHOLOGY, AGRICULTURAL EXPERIMENT STATION, UNIVERSITY OF FLORIDA, GAINESVILLE, FLORIDA

ALTERNARIA TENUIS THE CAUSE OF A TOMATO FRUIT ROT

L. P. McColloch

For many years confusion has surrounded the specific identity of the fungus that causes alternaria rot of tomato fruits. The disease has been ascribed to various species of Alternaria, and in the past it was also often referred to as macrosporium rot. In view of the present knowledge about the genus Alternaria a more definite classification of the causal fungus is now suggested.

Losses due to alternaria rot occur under certain conditions during the commercial handling and ripening of tomatoes harvested in the mature-green stage and also on vine-ripened fruits. Typical lesions are brown to black and may or may not have a definite margin, and the surface is flattened or slightly sunken. Decayed areas are firm and the decay extends into the flesh of the fruit and produces a dark brown to black, dry, core-like mass of decayed tissue. Dark gray mycelium is often found in the cavities in the decayed tissue. The surface of the spots frequently has a dense, velvety, olive-greenish appearance due to the mass development of spores.

Discussion of Nomenclature

The confusion regarding the cause of alternaria rot traces back to Galloway's description of black rot of tomato (2). Galloway was undoubtedly working with blossom-end rot, a physiological disease of tomato, and Alternaria was a secondary factor. He attributed this disease to Alternaria (Macrosporium) tomato (Cke.) Weber, but he labeled his illustrations Alternaria (Macrosporium) solani (Ell. & G. Martin) L. R. Jones & Grout. The spores illustrated do not fit either of these species, but resemble those of an Alternaria of the tenuis type. These errors were widely copied by other investigators, according to Weber (10).

In 1896 Jones and Grout (4), while making a study of Alternaria solani, isolated an Alternaria of the tenuis type, first from dead leaves of potatoes and later from decaying tomato fruits. They identified this fungus as A. fasciculata (Cke. & Ell.) L. R. Jones & Grout, erroneously making a new combination by including A. tomato. Failing to obtain infection when the fungus was inoculated into green tomatoes, they concluded that A. fasciculata was saprophytic.

For many years the confusion persisted concerning Alternaria tomato, the cause of nailhead spot; A. solani, known today as the cause of early blight of tomato plants and also of a dry, leathery spot on fruit in the field; and A. fasciculata.

In his review of the literature on Alternaria tomato and A. solani, Weber (10) amply clarified the systematic positions of these two species. His studies, however, did not directly concern the status of A. fasciculata other than to show that it is distinct from A. tomato.

The belief that Alternaria solani is one of the principal causes of decay of harvested tomato fruits has persisted to a certain extent. This is probably due to the facts that A. solani does cause a spot-type rot of tomatoes; that it has occasionally been isolated from tomato rots, together with the chain-forming tenuis type of Alternaria; and that it has been difficult to establish the pathogenicity of the chain-forming type, which has been considered more or less saprophytic since the work of Jones and Grout, who described A. fasciculata in 1897. The typical zoned, dry leathery lesions caused by A. solani should not, however, be confused with the alternaria rot discussed in this paper.

No publication pertaining to the Alternaria causing decay during the commercial handling and ripening of tomatoes harvested at the mature-green stage has been found, and the literature concerning the alternaria rot of tomatoes that occurs in the field or in laboratory tests on harvested fruits is not extensive.

Perhaps the first report of an Alternaria of the tenuis type causing decay of tomatoes was by McAlpine (6) in 1896. He designated the fungus as A. rugosa McAlp. In his studies of Alternaria published in 1944, Neergaard (7) considered A. rugosa identical with A. tenuis auct. sensu stricto.

In 1908 Pool (8) described a black rot of tomatoes and identified the causal fungus as Alternaria fasciculata. She stated that the description of A. rugosa agreed with that of her A. fasciculata with the exception of slight differences in color of the hyphae. The fungus reported by Pool caused decay of ripe tomatoes but not of green fruits.

Discussing plant diseases caused by the "blackening fungi", Bolle (1) stated in 1924 that the fungus so frequently found mixed with Alternaria solani on the leaf spots caused by the latter fungus is A. tenuis, with which this author considers A. fasciculata to be identical.

Warner (9) in 1936 attributed black rot of tomatoes to Alternaria sp. Infection was obtained on both green and ripe fruit. Warner reported that the spots ranged in size from "minute pinheads to areas extending completely across the surface of the fruit". Conidial measurements were 20.8 to 26.0 x 3.2 to 9.6 μ. These measurements, together with the illustrations, indicate that Warner's fungus would readily be considered A. tenuis under the present classification.

On the basis of Jones and Grout's account of Alternaria fasciculata and Bolle's studies on A. tenuis, Mason (5) applied the name A. tenuis to forms described as follows: Conidia in long chains, obclavate; the majority of spores 3 to 5 cross-septate (especially in culture) and falling within the limits 20 to 50 x 10 to 14 μ; conidia otherwise very polymorphous.

Wiltshire (11) suggested that, for convenience, fungi that fall within Mason's characterization, which is fundamentally based on Corda's concept, should be designated as Alternaria tenuis auct. As a result of the careful work of Mason (5), Wiltshire (11), Neergaard (7), and Groves and Skolko (3), A. tenuis auct. has become recognized as a broad species embracing many strains or forms previously accepted as species.

Present Investigation

Isolations from typical alternaria rot lesions over a period of years have uniformly yielded a chain-forming Alternaria of the tenuis type. This observation prompted the study herein reported. As it was considered desirable to examine this fungus from widely separated areas, isolates were made from affected tomatoes grown in California, Florida, Maryland, Texas, and Mexico. Single-spore cultures were made and carried in stock on Thaxter's agar. The isolates were grown on water agar for 3 to 10 days in preparation for measuring the conidia.

From two isolates from each locality 150 conidia taken at random were measured. Only the immature conidia with less than 3 cross septa at the terminal of the chain were rejected.

Considerable variation was found in the extent of sporulation of the isolates studied, and there was some variation in the development of the conidial chains, but all the chains were considered long. Table 1 shows the average size of the conidia and the extremes of size found in the 150 conidia measured. The conidia in the chains of some isolates were more uniform than these in others, and some isolates produced longer conidia than others. Strains producing extremely long, branched chains appeared to have the most variability in conidial length within the chain.

Table 1. Source of cultures and conidia measurements of Alternaria of the tenuis type isolated from tomato alternaria rot lesions.

Isolate No.	Source	Conidia measurements (microns) Average	Range
568	Beltsville, Md.	26.2 x 9.6	50.0 - 16.0 x 12.0 - 8.0
570	Beltsville, Md.	29.6 x 8.9	65.0 - 17.5 x 11.0 - 7.5
573	California	27.3 x 12.2	51.0 - 15.0 x 17.5 - 8.0
574	California	26.8 x 11.3	50.0 - 14.0 x 14.0 - 8.0
557	Florida	31.0 x 11.2	54.0 - 18.0 x 16.0 - 8.0
559	Florida	26.0 x 10.6	45.0 - 15.0 x 14.0 - 9.0
526	Mexico	23.9 x 9.7	45.0 - 15.0 x 12.5 - 7.5
542	Mexico	30.4 x 10.4	55.0 - 15.0 x 15.0 - 8.0
571	Texas	32.3 x 12.3	55.0 - 20.0 x 20.0 - 9.0
572	Texas	29.8 x 12.3	45.0 - 17.5 x 19.0 - 9.0

Except for the extremes in length and diameter of a few of the conidia, the average and extremes for all isolates readily come within Mason's specifications. It therefore seems that the isolates studied are strains of Alternaria tenuis auct., and that this species is responsible for alternaria rot of tomato fruits.

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DIVISION OF FRUIT AND VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE

BOTRYTIS LEAF SPOT OF APPLE IN NORTH CAROLINA

C. N. Clayton

A necrotic blotch or spot of apple leaves was very common in many orchards in Wilkes and Alexander Counties in May of 1950. The somewhat irregularly shaped spots varied from a few millimeters to more than 2 cm. in diameter. Usually an infected dead blossom part, such as a petal or anther, was adhering to the affected tissue. A species of Botrytis was invariably found sporulating on the necrotic leaf and blossom tissues.

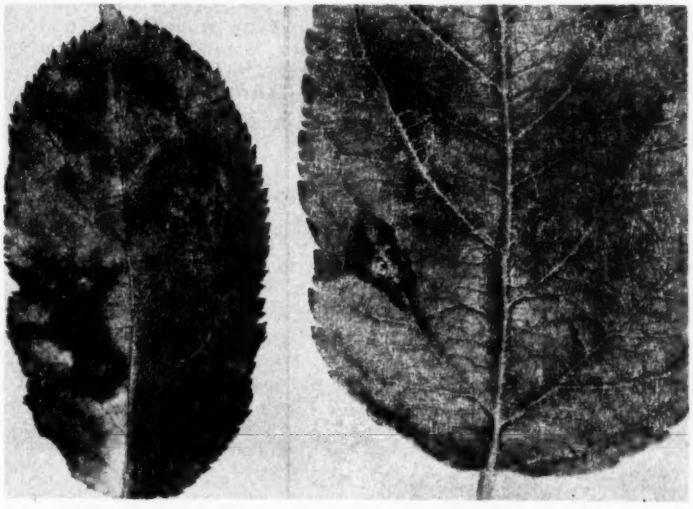

FIGURE 1. Botrytis leaf spot of apple leaves. Note infected petals in contact with leaf on left and infected anther on leaf on right.

The development of the disease was favored by moisture and temperature conditions subsequent to full bloom. Just as full bloom of apple occurred, on April 28 and 29, the maximum and minimum temperatures were close to 83° and 58° F., respectively. This warm weather resulted in the blossoms reaching the petal-fall stage just as the five-day rainy period of April 30 to May 4 started. During this period the minimum night temperatures were between 56° and 59° F. and the maximum day temperatures were between 70° and 77° F. Evidently, infected flower parts after detachment were blown to the leaves, where infection occurred. Sporulation of the fungus was abundant by the end of another five-day rainy cloudy period of May 11 to 15. By May 18 some leaves were chlorotic and were being abscised. This was particularly the case on leaves with lesions involving the midrib or larger veins.

The actual defoliation that resulted from the spotting of the leaves probably was 5 percent or less and resulted in neglibible damage to the crop.

The Botrytis was readily isolated from affected leaf parts. Mature healthy excised apple leaves were inoculated by placing a small amount of mycelium on agar onto the surface of leaves on wet paper in Petri plates. After about a week typical large necrotic blotches or spots surrounded the points of inoculation. From such leaf tissue the Botrytis was easily re-isolated. On potato dextrose agar black sclerotia were readily produced.

The relative amounts of the disease in one orchard on non-sprayed trees and on those sprayed three times (pre-pink, April 11; pink, April 20; and calyx, May 6) with 341-SC, Phygon-XL, Flotation Sulfur paste, liquid lime-sulfur, and Tag Fungicide No. 331 were determined on May 18. The number of leaves affected was determined by examining 200 leaves at random on each of three Red Delicious and three Stayman trees for each spray program. The average percentage of infected leaves on non-sprayed trees was 7.5, while on sprayed trees it ranged from 2.4 to 3.6.

PLANT PATHOLOGY LABORATORIES, NORTH CAROLINA STATE COLLEGE, RALEIGH

STRAWBERRY ROOT-ROT IN MINNESOTA[1]

E. Butler and T. H. King

Black root-rot is recognized as an important factor in strawberry degeneration in Minnesota Many investigators (1) (2) (3) (4) have reported this complex disease, but it has not been studied extensively in Minnesota. Preliminary work in 1949 included field surveys, isolation of fungi from diseased roots, and testing pathogenicity of various fungus isolates.

The disease is characterized by the appearance of black cortical lesions any place on the root. These increase in number and size, eventually resulting in blackening and death of the entire root cortex. Fibrous roots are conspicuously absent. The root system of a plant may become completely rotted, but disease development generally proceeds more slowly than the formation of new adventitious or secondary roots. Hence, plants with extensively damaged root systems may appear healthy for more than one season. Under drought conditions, however, infected plants may wilt and die. Dwarfed plants with small leaves as described by Miller (2) and others (1) (3) (4) were found infrequently, but many diseased plants had red-brown to red leaves.

FIELD OBSERVATIONS. Eleven fields in Ramsey and Olmstead Counties were examined from July to November inclusive. Root rot was especially severe in poorly drained soils with a high pH. In general, 80 to 100 percent of the spring-set plants had roots with black lesions by July. The number of runner plants affected seemed to vary with pH and drainage. In two fields with alkaline soils and obviously poor drainage, 80 to 90 percent of the runner plant roots had numerous black lesions when three to four inches long. There was no correlation between incidence of root rot and soil type, variety of strawberry, or previous crop growth. In replicated plots of 75 varieties grown at Rochester, Minnesota, where a previous crop of soybeans had been turned under, root rot was noticeably less damaging than in the average fields except for variety Corvallis, which had severe root rot symptoms. No plants were found with symptoms of the red stele disease.

FUNGI ASSOCIATED WITH ROOT LESIONS. Diseased material was obtained from 20 fields in Olmstead, Ramsey, St. Louis, and Hennepin Counties. Root sections containing black sunken lesions or black girdled areas were treated five minutes with 0.5% sodium hypochlorite and plated on prune agar (pH 4.2-4.5). Isolations were made from varieties Burgundy, Dunlap, Premier, Robinson, Corvallis, and the ever-bearing varieties Gem and Redrich.

The following organisms were associated with diseased roots: Fusarium spp., 50[2], Penicillium spp., 11; Coniothyrium fuckelii, 7; Chaetomium globosum, 6; Hainesia lythri, 5; Rhizoctonia solani, 4; Rhizoctonia spp. (orchid type), 0.3; Trichoderma spp., 3; Aspergillus spp., 3; Mucor spp., 3; Rhizopus spp., 3; Zygorhynchus sp., 0.8; Robillardia sp., 0.5; Gliocladium roseum, 0.5; Phoma sp., 0.2; Alternaria sp., 0.2; Ramularia sp., 0.2; Verticillium sp., 0.2; Mycogone sp., 0.2; and unidentified isolates, 4.5.

Hainesia lythri was first isolated in late October from the varieties Gem and Corvallis. Other organisms encountered were not seasonal in occurrence.

In fields where several varieties were grown, diseased roots had a similar fungus flora. Fusarium spp. were the only fungi isolated consistently from all varieties. In November, Fusarium spp. were isolated from runner-plant roots four to eight inches long with no observable lesions. Plants with roots two to three inches long were usually free from fungi.

PATHOGENICITY OF SELECTED FUNGUS ISOLATES. A hybrid selection, Minn. 322-2, moderately susceptible to root rot in field plots, was used for pathogenicity tests. Plants were grown in initially sterile sand until the roots were three to four inches long and then used to test the isolates. Inoculum was increased on oats and added to a sandy soil, pH 6.0, at 60-65° F. and 80-85° F. After 40 days in the infested soil the roots were examined for lesions.

Hainesia lythri, Coniothyrium fuckelli and Rhizoctonia solani were pathogenic, while Mucor sp., Chaetomium globosum, Robillardia sp., and Verticillium sp. did not cause root lesions. In two tests approximately 75 percent of the adventitious roots of the plants from soil infested with Hainesia lythri developed lesions at 80-85° F. However, at 60-65° F. only 5 percent of the roots were diseased. Rhizoctonia solani and Coniothyrium fuckelii caused lesions

[1]Paper No. 2285, Scientific Journal Series article, Minnesota Agricultural Experiment Station.
[2]Number following name of organism indicates percentage of total number of isolates.

on approximately 50 percent of the roots. Hainesia lythri was the most virulent of the organisms tested. Although root systems were extensively damaged (numerous lesions per root), in no instance were disease symptoms apparent in the above-ground plant parts. Lesions were also present on the roots of some of the control plants. The fungi associated with these lesions were mainly Trichoderma spp. and Penicillium spp. Unfortunately studies were temporarily discontinued before the various isolates of Fusarium could be tested for pathogenicity.

The results indicate that many saprophytic and weakly parasitic fungi are associated with strawberry root rot. The combined effect or interaction of these fungi with other environmental factors remains to be investigated.

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DIVISION OF PLANT PATHOLOGY AND AGRICULTURAL BOTANY, UNIVERSITY OF MINNESOTA, ST. PAUL

OBSERVATIONS ON THE PATHOGENICITY OF RHIZOCTONIA SOLANI ON BENT GRASSES [1]

John B. Rowell [2]

Certain observations have been made in the course of studies on the control of turf diseases in Rhode Island which introduce new concepts in the epidemiology of brown patch of bent grasses. This disease, which is caused by Rhizoctonia solani, is an important hot weather pest for greenkeepers throughout the northern States, where it markedly disfigures putting greens during the summer months. The following studies on the pathogenicity of R. solani on bent grasses used in greens have indicated that the wounds produced by the frequent mowing of the turf and the guttation drops produced on these surfaces are important factors in the development of the disease that have not been fully considered in earlier investigations.

Turf workers have commonly observed that brown patch appears most frequently and severely on the short cut turf of the green. The same variety of grass in the surrounding apron may be entirely free of the disease. The two areas differ in length of cut; the green proper being cut to 1/4 inch by two to four mowings per week, while the aprons are cut less frequently to a 3/4 inch height. This is good circumstantial evidence of the importance of closeness and frequency of cut to the pathogenicity of Rhizoctonia solani on Agrostis spp. However, the role of wounding in this disease has never been indicated in previous literature.

The pathogen spreads within a diseased area by mycelial growth from a central point, producing roughly circular, wilted, brown areas from one inch to several feet in diameter depending, among other factors, on the duration of the weather conditions favorable for the disease development. At the margin of the area a typical "smoke ring" of grayish-black mycelium from 1/2 to 1 1/2 inches wide delimits the area where the fungus is actively growing. In this area the stages of symptom development of the disease on the grass plant can be observed as the leaves become first water-soaked, wilt, then blacken, collapse, dry and finally become light brown in color. The fact that infection frequently occurs through the mowing wounds is shown by close examination of grass blades at the margin of an actively spreading diseased area of brown patch. The tips of the grass blades are first affected by the disease, which later progresses downwards towards the crown of the plant. Generally, under Rhode Island conditions, according to Howard et al. (5), the proper environmental conditions do not persist over a period of time sufficiently long for the disease to kill the crown and roots and thus permanently damage the turf. This is explained in part by the facts that infection is initiated at the tips of the leaves and that the pathogenicity of R. solani on bent grass is inactivated by minimum temperatures dropping below 70° F. as reported by Dickinson (4) and Dahl (3). The cooler temperatures check the further spread of the pathogen to non-diseased turf areas, and also halt the growth of the parasitic mycelium within the host leaves. As the leaf blades grow in length outwards from the crown meristems, the diseased portion of the leaf is pushed upwards and is cut off by the frequent mowing of such turf so that all evidence of the disease soon disappears unless another active period occurs.

Inoculations of turf grasses were made in the greenhouse to study the disease critically. Sod sections were removed from established four-year old Congressional creeping bent (Agrostis palustris) and transplanted into flats in the greenhouse. Young turf of Colonial bent grass (A. tenuis) was grown from seed in steam sterilized soil in 4-inch pots. The isolate of Rhizoctonia solani used for inoculations was isolated from brown patch occurring on R. I. Colonial bent at Kingston, Rhode Island, in August, 1947. The grass was inoculated by spraying the turf with a suspension prepared by maceration of the mycelium from a young culture of the pathogen grown in potato-dextrose broth. Then the turf was maintained for 48 hours in a moisture chamber at a temperature of 75° to 85° F. Repeated inoculation trials with uncut grasses of the two types failed to produce typical brown patch. A few infected leaves could always be found, but extensive spreading of infective hyphae to adjacent leaves never occurred. However, when either turf was cut short and inoculated in the same manner, the mycelium of R. solani rapidly overgrew the entire turf mass, producing severe brown patch symptoms within 18 hours.

The turf was allowed to recover from this initial brown patch attack by growth in a greenhouse maintained at 65-70° F. During this period the turf was cut at weekly intervals. Three weeks later, turf of each type was divided into two lots; one of which was cut to approximately

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[1] Contribution No. 789 from the Rhode Island Agricultural Experiment Station.

[2] Former Assistant Research Professor, Rhode Island State College, Kingston, Rhode Island, at present Research Associate, University of Minnesota, St. Paul, Minnesota.

1/2 inch height, and the second remained uncut with the leaves 2 to 3 inches in length. The turf was again inoculated with macerated mycelium from potato-dextrose broth cultures of R. solani and incubated as previously outlined. Severe, typical brown patch developed on all the cut grasses, while only spotty infection occurred on the uncut grasses. One additional test illustrated the importance of mower wounding to the development of the disease. Young Colonial bent seedlings were grown in steam sterilized soil in pots. When the grass leaves were approximately 2 to 3 inches long, the grass in one half of the pot was cut to 1/2 inch height and the remainder left uncut. After inoculation and incubation, the disease again developed severely on the cut leaves and only sparsely on the uncut portions; the extensive pathogenic mycelium on the cut grass failed to vigorously attack the uncut blades.

The infection trials indicated that two factors, wounds and guttation water, are probably important in the more vigorous development of R. solani in attacking freshly cut turf. The fungus is known to attack many plants by entering through stomata, and Dickinson (4) has indicated that this is the mode of attack in turf grasses. However, wounds appear to have the major role as infection courts in short-cut bent turf, as indicated by the more ready disease development on cut leaves and also by the observation that infection originates at the tip of the blade and proceeds downwards toward the crown on the plant. Only in the long uncut leaves have infrequent infections been found in the center of the blade.

The accumulation of the liquid at the tips of cut grass blades has characteristics of both the processes of guttation and bleeding. The exudation of water from an injured surface rather than a hydathode is characteristic of the latter process. However, in turf the drops are not continuously produced from the wounds, as in bleeding, but occur mainly under the conditions favorable for guttation in which the roots rapidly absorb water and the rate of transpiration is reduced. As the production of liquid drops at the tips of either cut or uncut grass blades is commonly considered to be guttation without regarding theoretical implications, this terminology will be followed.

Guttation drops appear to be a second important factor in brown patch epidemiology by contributing to the rapid saprophytic growth of the pathogen in the infection court area. Such drops have been found to appear on turf at Kingston by 9 P.M. and persist until 11 A.M. of the following day, during the hot and humid conditions known as "brown patch weather". Under foggy conditions the drops may persist throughout the daylight hours. The occurrence of dissolved salts and organic materials in guttation water make the drops an ideal culture medium for saprophytic development. Hyphae of the pathogen have been observed bridging from guttation drop to guttation drop at the tips of the blades both in greenhouse and field infections, with masses of mycelia growing within the drops.

All severe brown patch infections observed in these studies occurred when the turf was moist with guttation drops. The disease was not observed to develop during rain storms. This may be explained by the failure of temperatures to be favorable during the rain. However, the disease was not observed to develop during overhead irrigation of the turf with favorable temperatures. A check of the temperature of the irrigation water at the outlet showed its temperature to be 56° F., but a thermometer placed on the surface of the turf in the spray recorded the same temperature (85° F.) as one similarly placed outside the irrigated area. Attempts to produce the disease experimentally in turf plots by spreading inoculum of R. solani grown on a steam-sterilized wheat-oats substratum and watering with overhead irrigation were unsuccessful. However, two epidemics of the disease were produced on plots of R. I. Colonial and Toronto creeping bent grasses when the inoculated turf was covered with burlap strips that were kept sufficiently moist so the grass was maintained continuously in a humid atmosphere favoring the development and persistence of guttation drops.

Greenkeepers have long known that poling and hosing the greens early in the morning, both common practices in turf management for removing "dew", are of definite value in minimizing the development of the disease. Hosing is believed by these men to be effective in breaking up the mycelia. Dickinson (4) demonstrated that the short germination hyphae of R. solani sclerotia are easily destroyed by mechanical damage and believes this explains the protective action of poling. However, in the present trials the disease was readily produced by an inoculum consisting of mycelial fragments produced by maceration of a broth culture in a Waring blendor. When the disease is active in turf the practice of poling and hosing would thus appear to aid in disseminating the pathogen throughout the area rather than to destroy the mycelia. However, the protective action of these control measures can readily be explained by the dispersion and subsequent rapid drying of the moisture and nutrients of the guttation drops, which can no longer give impetus to the saprophytic growth of the pathogen at the infection court. This view could be tested by spraying the turf with various surface tension depressants which could prevent the

retention of plant exudates. For example, Isonol DL 1 (dilauryl dimethyl ammonium bromide) was found by Keil and Howard (6) to make the poling of greens unnecessary for at least five mornings after application because "dew" (guttated water?) failed to stick to the grass blades.

The development of brown patch during periods of excessive guttation may complicate fungicidal control measures. Curtis (2) has previously demonstrated the role of the solutes of guttated water in activating certain copper-containing fungicides. Laboratory studies were initiated to determine the effects of possible constituents of guttated water on turf fungicides in an attempt to elucidate some of the inconsistencies in the performance of these materials as protectants against the brown patch disease. The most striking results of these preliminary studies was the inactivation in various degrees of some fungicides by glutathione. The phenyl mercury compounds were most markedly affected, indicating that metabolites with sulfhydryl groups in guttation water may influence field performance of these materials.

One further phase of the disease was investigated. The symptoms exhibited by diseased grass blades strongly suggested a toxic effect by the pathogen, i.e., the blade first appears water soaked and wilts, changing to a bluish-green to black followed by collapsing, drying and browning. Toxic substances produced during the saprophytic growth of the pathogen in the guttation drop could be drawn into the plants as demonstrated by Curtis (1). Various attempts to introduce cell-free filtrates of different liquid media on which R. solani had been grown into the tops of grass blades failed to reproduce any disease symptoms. These negative results were of questionable significance, however, as the amount of liquid that penetrated the leaf tissues was unknown.

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RHODE ISLAND STATE COLLEGE, KINGSTON

OBSERVATIONS ON REPEATED APPLICATIONS OF D-D (DICHLOROPROPENE-DICHLOROPROPANE)

C. W. McBeth[1]

One of the first questions that arose with the advent of field scale soil fumigation with D-D* was whether repeated applications of this material would result in the accumulation of residues toxic to plant growth. It has been recognized from the beginning that D-D is highly toxic to higher plants as well as nematodes, insects and to a limited extent, fungi. Therefore, a reasonable time must elapse between the application of D-D and the planting of the crop. It has also been known that this interval between application and planting is influenced by several factors such as dosage, soil temperature, soil moisture, soil type, and weather conditions immediately following application.

Because D-D is a comparatively new material and has not been in commercial use for a long period of time, the residual effect from repeated applications has not been determined. To the best of the writer's knowledge, the only careful study of this problem, under controlled experimental conditions, was made by Shaw[2] as part of his doctoral thesis. In this laboratory work Shaw found that the movement of D-D as a liquid occurs in the first two hours and that any further movement is probably in the form of a vapor. The rate at which D-D diffuses from a laboratory soil sample was determined by measuring the daily weight loss of a sample containing it. Shaw found that 60 percent of the D-D is lost after one week: 88 percent after two weeks and 90 percent after three weeks. Two months later there remained a non-volatile residue approximating 8 percent of the original injection. The heavy tarry fraction appeared to remain in the soil indefinitely.

Assuming that Shaw's results can be applied to commercial application of D-D under field conditions, 8 percent of a 200-pound per acre application, or 16 pounds, would remain as a residue in the soil. This 16 pounds per acre would amount to approximately 4 to 5 parts per million in the first foot of soil, which does not appear to be enough to cause concern. There is the possibility, however, that under repeated application the residue might accumulate to levels detrimental to plant growth. The work summarized in this paper was initiated with this in mind.

Methods and Results

An area 200 feet x 180 feet was used for this study. The soil type was Fresno sandy loam with a moisture equivalent of 10 percent. This area was divided into three equal strips or plots 60 feet x 200 feet. Plots were not replicated, as it was felt that for this type of experiment one large plot was much more desirable than several small ones. One plot was left untreated for a five-year period and the other two were treated by commercial applicator each year, one at 200 pounds and the other at 400 pounds per acre. Each year a variety of crops has been grown on each plot such as melons, beans, peas, tomatoes, and tobacco. Although actual yields have not been taken on these plots, the general appearance and productivity of the treated plots has been superior to the check plot, the most thrifty plants being in the 400-pound per acre plot. It is felt that any residual toxicity of any importance should be obvious to visual observation. Root knot nematode examinations were made after the fourth and fifth treatments. No infection was found in the treated plots, while the tomatoes, melons, and tobacco showed 100 percent infection in the check area.

Photographs were taken at the end of the fifth growing season to illustrate the lack of any indication of toxic residues in the treated areas. Figure 1 is from the 400-pound treatment showing melons on right, one row of tobacco next, three rows of tomatoes and several rows of cowpeas on the left. Figure 2 is a photograph of the untreated area, melons on the right, one row of tobacco, three rows of tomatoes and peas on the far left.

By comparing Figure 2 and Figure 1, it becomes very evident that the growth of all crops is more vigorous in Figure 1, which has had five yearly applications of 400 pounds of D-D per acre.

As a further comparison of the growth response, a representative tomato plant was dug from the check and from the 400-pound per acre plot and photographed. This is illustrated in Figure 3, check plant on right and treated plant on left. The difference in root and top growth in these

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*Trade-mark registered U. S. Patent Office.

[1]Nematologist, Shell Oil Company Agricultural Research Laboratory, Modesto, California.

[2]The effect of Soil Sterilant (Dichloropropene-Dichloropropane mixture) on the Chemical, Physical, and Microbiological Properties of Desert Soils. Department of Agricultural Chemistry and Soils, University of Arizona. May, 1949.

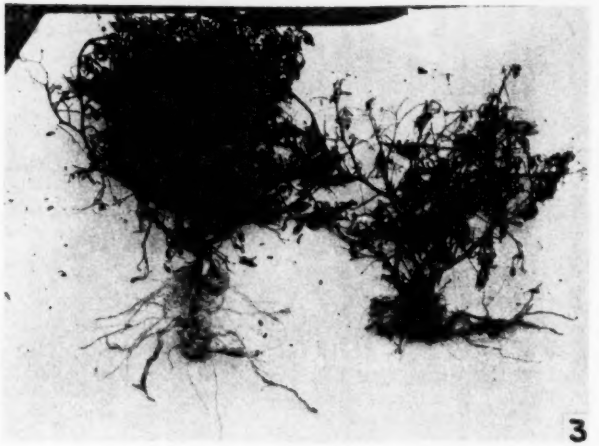


Figure 1. D-D, 400 pounds per acre each year for five consecutive years. Melons at right, one row of tobacco, three rows of tomatoes, and several rows of cowpeas at left. Check plot of melons and tobacco at extreme left.

Figure 2. Untreated plot. Melons at right, one row of tobacco, three rows of tomatoes, and several rows of cowpeas on the left. Note bare ground visible between tomato plants and in melon area.

Figure 3. Presesentative tomato plants. Right plant from untreated area and left from D-D 400 pounds each year after fifth year of consecutive treatments. Plant from untreated area heavily infected with root knot nematode.

two plants is very striking.

Conclusions

D-D applied annually at up to 400 pounds per acre has given no indications of a residue accumulating in the soil which is toxic to plant growth. Larger and healthier tomato, tobacco, melon, and cowpea plants have been grown on an area treated with 400 pounds of D-D each year for five consecutive years than on adjacent, untreated area. This experiment does not rule out the possibility that a retarding effect may be present but is masked by a nematode infection; however, it is quite apparent that retardation of growth from nematodes is more severe than from a toxic residue. It also follows that the chemical application would not be made in most cases unless nematodes or some other parasites were present.

SHELL OIL COMPANY, MODESTO, CALIFORNIA

UNUSUAL RECORDS OF PLANT DISEASE OCCURRENCE

UNUSUAL OCCURRENCE OF ASCOSPORIC INFECTION OF TOMATOES BY SCLEROTINIA SCLEROTIORUM

By Roy Bardin

A field of approximately twenty acres of several varieties of tomatoes grown for seed production in the Salinas area of Monterey County was found generally infected by Sclerotinia sclerotiorum early in October 1950. A close examination of many plants revealed that infection had occurred some time prior to the discovery of the disease. Since the infections of nearly all diseased plants were not contiguous with the roots or crowns, nor with plant parts touching the soil, it is believed that the source of infection was largely that of ascospores. Partially developed fruit and the pith cavities of the blighted branches carried an abundance of mature sclerotia. Symptoms consisted of whitish lesions scattered over twigs, stems, and on the fruit, characteristic of this fungus on other hosts. Heavy November rains later assisted in a general infection of Botrytis sp. on these dead plant parts.

To the knowledge of the writer no such large scale ascospore infection of a commercial tomato planting at this time of the year has been previously noted in the region. It has been reasoned that in this instance an ascospore discharge probably occurred in a frequently irrigated permanent pasture some one-fourth to one-half miles distant from the tomato field. Both the tomato field and an intervening field of dry beans were clean-cultivated, were very free of weed growth, and had been frequently irrigated.

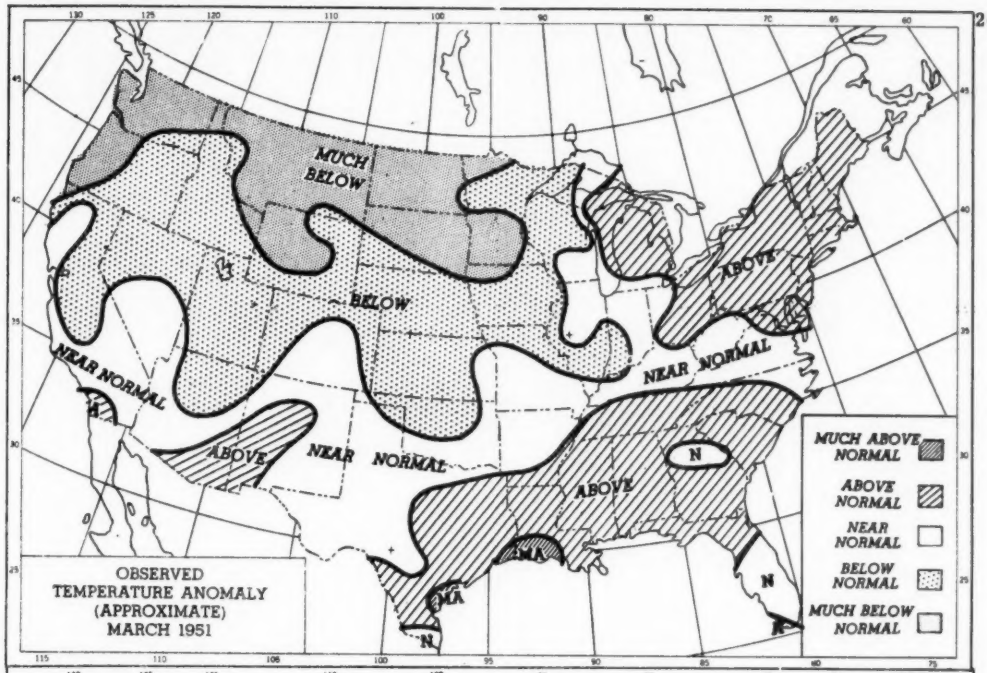
It is believed that these fields hardly could have supported necessary apothecia for the discharge of ascospores at this time of year since no rain fell during this period. The irrigated permanent pasture land on the other hand was known to carry endemic S. sclerotiorum infection and it seems highly probable that under the ideal conditions of temperature, moisture, and protection afforded there that a good supply of apothecia may have been produced. Other tomato fields two to three miles distant as well as fields distributed in the valley, suffered no such infection.

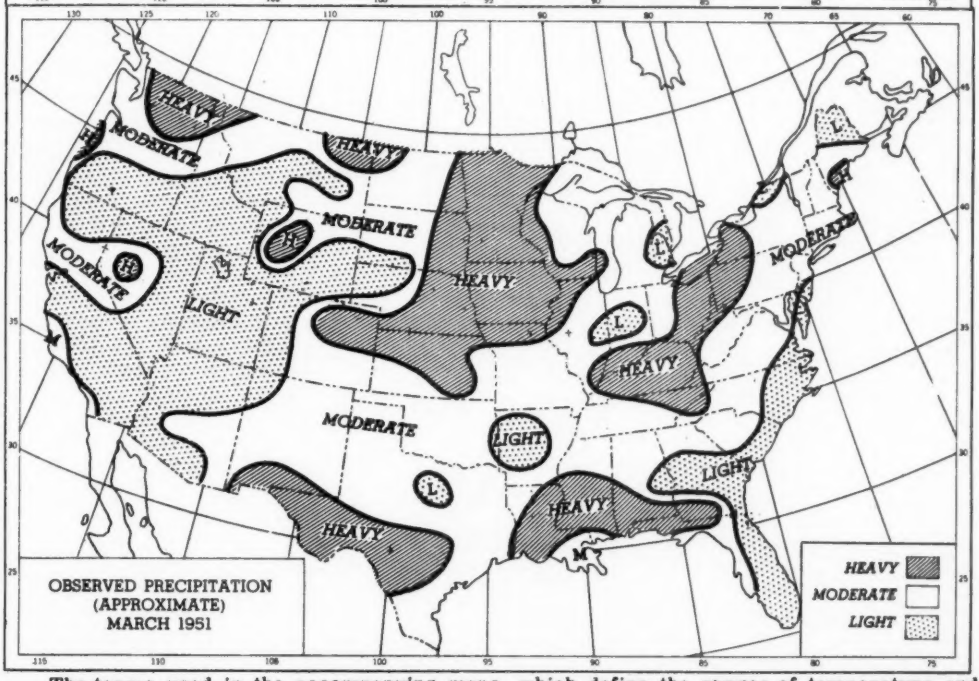
The evidence would indicate that tomato plants grown in the Salinas Valley are subject to damage from ascospore infection by the fungus under some conditions prevailing in the late summer or fall provided that adequate inoculum be present. The uniqueness of the infection reported here ties in with the observation that apothecia are usually absent in the area until after the fall rains begin.

MONTEREY COUNTY DEPARTMENT OF AGRICULTURE, SALINAS, CALIFORNIA

A CORRECTION

Our attention has been called to an error in reference citation in the April issue, page 218. The date for reference number 1 should be 1942, not 1949.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.